

2. Kerr F, Kenoyer G, Bilitch M: Quinidine overdose—Neurological and cardiovascular toxicity in a normal person. *Br Heart J* 33:629-631, Jul 1971
3. Luchi RJ, Helwig J, Conn HL: Quinidine toxicity and its treatment. *Am Heart J* 65:340, 1963
4. Conn HL, Luchi RJ: Some quantitative aspects of the binding of quinidine and related quinoline compounds by human serum albumin. *J Clin Invest* 40:509, 1961
5. Shub C, Gau GT, Sidell PM, et al: The management of acute quinidine intoxication. *Chest* 73:173, 1978
6. Bismuth C, Pebay-Peyroula F, Frejaville J: Traitement actuel des cardiopathies toxiques. *Concours Med* 91 (Suppl) 21:4453-4470, 1969
7. Brown T: Tricyclic antidepressant overdose: Experimental studies on the management of circulatory complications. *Clin Toxicol* 9:255-272, 1976
8. Biggs JT: Clinical pharmacology and toxicology of antidepressants. *Hosp Pract*, Feb 1973, pp 79-84
9. Burckhardt D, Raeder E, Muller V, et al: Cardiovascular effects of tricyclic and tetracyclic antidepressants. *JAMA* 239: 213-216, 1978
10. Coté M, Elias G: Le propranolol dans les arythmies cardiaques par intoxication à l'imipramine (Tofranil) chez l'enfant. *Union Med Can* 103:1223-1225, 1974
11. Callahan M: Tricyclic antidepressant overdose. *JACEP* 8: 413-425, Oct 1979
12. Hagerman GA, Hanashiro PK: Reversal of tricyclic-antidepressant-induced cardiac conduction abnormalities by phenytoin. *Ann Emerg Med* 10:82-86, 1981

Ascorbic Acid and Nutrition

TO THE EDITOR: I am writing about the article by Richard Vilter, MD, "Nutritional Aspects of Ascorbic Acid: Uses and Abuses" in the December 1980 issue.¹ It is extremely well written and will certainly serve as a classic in the field, as our medical attention is drawn more and more to the apparent important aspects of nutrition and nutritional deficiency state. I would take issue with only a few points in the article.

First, one need only assess the incidence of false teeth in our country to ascertain whether Dr. Irwin Stone's hypothesis of very prevalent chronic subclinical scurvy has any merit.

Also, I reviewed references 73, 74, 75, 76, 78 and 80 from the article. In reading these review articles and original works, it becomes very evident that no type of blood testing to determine the at-risk population among those given vitamin C was used. Truly this is a breach of good scientific study. Until a reliable method of determining accurately serum vitamin C levels and pertinent factors is used, we will not be able to determine an at-risk group of persons, nor will we be able to assess accurately the efficacy of any dose of ascorbic acid, by whatever route applied, in the treatment of common colds.

To date we have tested 80 patients with a request for serum vitamin C levels. This has usually given us the reduced vitamin C, or ascorbic acid, content of the blood. I have found no specific trend according to this technique. My practice is composed mainly of chronic debilitated orthopedic-pain patients.

Recently, we tested 30 patients according to the technique indicated by Irwin Stone.² According to the "morbidity index" that he has defined, most of our patients have demonstrated values in the range of the "survived" or "convalescent" serum levels. The results that we have obtained seem to corroborate, on a preliminary basis, the fact that our patients are indeed clinically debilitated. There are other nutritional factors that we are presently assessing. It is my impression that Dr. Stone's morbidity index at least gives us a standard from which to compare possibly normal and abnormal patients. I am sure that this represents merely a step in the right direction, however. I believe that other measurements will be defined in the future so that we may be able to determine more accurately the specific group at risk from the low vitamin C levels in the blood.

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REFERENCES

1. Vilter RW: Nutritional aspects of ascorbic acid: Uses and abuses (Nutrition in Medicine). *West J Med* 133:485-492, Dec 1980
2. Stone I: The Healing Factor—Vitamin C Against Disease. New York, Grosset and Dunlap, Publisher, 1972

Hypokalemia in the Syndrome of Inappropriate Secretion of Antidiuretic Hormone

TO THE EDITOR: The cause of hypourcemia¹ and hypouricemia² in the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) has been thought to be increased urinary losses of the above metabolites. I wish to report the association between hypokalemia and severe hyponatremia where the hypokalemia probably reflected increased urinary losses.

Eight cases were studied in which hyponatremia of less than 110 mEq per liter developed during patients' hospital stays and in which the criteria for the diagnosis of SIADH³ also were fulfilled. Four patients had malignant lesions (lung two, brain one, breast one), two had tuberculosis, one patient presented with pneumonia and the last patient had a cerebrovascular accident. All patients received at least five liters of 5 percent glucose in water intravenously during the first seven-day period in hospital. Serum sodium and potassium levels were measured at least four times from the time of admission to the time the serum sodium value fell below 110 mEq per liter. The results showing the change in electrolyte concen-